Amendments to the Specification:

Please replace the paragraph at page 1, from line 10 through line 15, with the following paragraph:

--This application is a divisional of U.S. Application Serial No. 09/938,703, filed August 24, 2001, which is a divisional of U.S. Application Serial No. 09/626,939, filed July 27, 2000, which is a divisional of U.S. Application Serial No. 08/833,752, filed April 9, 1997, which is a continuation of U.S. Application Serial No. 08/810,028, filed March 3, 1997~~March 4, 1997~~, which claims the benefit of EP 96870021.1, filed March 1, 1996 and EP 96870102.9, filed August 6, 1996. The entire teachings of the above applications are incorporated herein by reference.--

Please replace the Brief Description of Figure 1, on page 14, line 6 with:

-- Figure 1 ~~Represents the primary structure of the peptides according to the invention~~ shows the nucleic acid and amino acid sequences of the invention. Figure 1A-1 and 1A-2 show the nucleic acid and amino acid sequence of SEQ ID Nos 1 and 4, respectively. Figure 1B-1, 1B-2, and 1B-3 show the nucleic acid and amino acid sequence of SEQ ID Nos 2 and 5, respectively. Figure 1D-1 to 1D-3 show the nucleic acid and amino acid sequence of SEQ ID Nos. 3 and 6, respectively.--

Please replace the Brief Description of Figure 2, on page 14, lines 8-11, with:

-- Figure 2 represents the amino acids sequence of the active human CCR5 chemokine receptor (SEQ ID NO: 5) according to the invention aligned with that of the human CCR1 (SEQ ID NO: 9), CCR2b (SEQ ID NO: 7), CCR3 (SEQ ID NO: 8), and CCR4 (SEQ ID NO: 10) receptors. Amino acids identical with the active CCR5 sequence are boxed.--

Please replace the Brief Description of Figure 6, on page 14, line 18, with:

-- Figure 6 represents the structure of the mutant form of human CCR5 receptor. Figure 6B shows the wild type amino acid sequence (SEQ ID NO: 11), and the location of the 32 base deletion mutation in the nucleic acid (SEQ ID NO: 12) and amino acid sequences (SEQ ID NO: 13). --

On pages 22-23, please replace the paragraph, extending from page 22, line 25 through page 23, line 31, with the following replacement paragraph;

-- It is known that some individuals remain uninfected despite repeated exposure to HIV-1 [55, 56, 51]. A proportion of these exposed-uninfected individuals results from the relatively low risk of contamination after a single contact with the virus, but it has been postulated that truly resistant individuals do exist. In fact, $CD4^+$ lymphocytes isolated from exposed-uninfected individuals are highly resistant to infection by primary M-tropic, but not T-tropic HIV-1 strains. Also, peripheral blood mononuclear cells (PBMC) from different donors are not infected equally with various HIV-1 strains [57-59]. Given the key role played by CCR5 in the fusion event that mediates infection by M-tropic viruses, it is postulated that variants of CCR5 could be responsible for the relative or absolute resistance to HIV-1 infection exhibited by some individuals, and possibly for the variability of disease progression in infected patients [62]. The Inventors selected three HIV-1 infected patients known to be slow progressors, and four seronegative individuals as controls; the full coding region of their CCR5 gene was amplified by PCR and sequenced. Unexpectedly, one of the slow progressors, but also two of the uninfected controls, exhibited heterozygosity at the CCR5 locus for a biallelic polymorphism. The frequent allele corresponded to the published CCR5 sequence, while the minor one displayed a 32 bp deletion within the coding sequence, in a region corresponding to the second extracellular loop of the receptor (FIG. 6). Figure 6 shows the structure of the mutant form of human CC-chemokine receptor 5. Figure 6a shows the amino acid sequence of the nonfunctional ΔCCR5 protein is represented. The transmembrane organization is given by analogy with the predicted transmembrane structure of the wild-type CCR5. Amino acids represented in black correspond to unnatural residues resulting from the frame shift caused by the deletion. The mutant protein lacks the last three transmembrane segments of CCR5, as well as the regions involved in G protein-coupling. Figure 6b shows the nucleotide sequence of the CCR5 gene surrounding the deleted region, and translation into the normal receptor (top) or the truncated mutant (CCR5, bottom). The 10-bp direct repeat is represented in italics. The full size coding region of the CCR5 gene was amplified by PCR, using 5'

TCGAGGATCCAAGATGGATTATCAAGT-3' (SEQ ID NO: 14) and 5'-CTGATCTAGAGCCATGTGCACAACTCT-3' (SEQ ID NO: 15) as forward and reverse primers' respectively. The PCR products were sequenced on both strands using the same oligonucleotides as primers, as well as internal primers, and fluorochrome-labelled didcoxynucleotides as terminators. The sequencing products were run on an Applied Biosystem sequencer, and ambiguous positions were searched along the coding sequence. When the presence of a deletion was suspected from direct sequencing, the PCR products were cloned after restriction with *BamHI* and *XbaI* endonucleases into pcDNA3. Several clones were sequenced to confirm the deletion. The deletion was identical in three unrelated individuals investigated by sequencing.--

On page 25, please replace the paragraph extending from page 30, line 8 through line 26, with the following replacement paragraph:

--The figure 8 represents genotyping of individuals by PCR and segregation of the CCR5 alleles in CEPH families. Figure 8a shows autoradiography illustrating the pattern resulting from PCR amplification and EcoRI cleavage for individuals homozygous for the wild-type ***CCR5*** allele (CCR5/CCR5), the null ΔCCR5 allele (ΔCCR5/ΔCCR5)- , and for heterozygotes (CCR5/ ΔCCR5). A 735 bp PCR product is cleaved into a common band of 332 bp for both alleles, and into 403 and 371 bp bands for the wild-type and mutant alleles, respectively. Figure 8b shows segregation of the CCR5 alleles in two informative families of the CEPH. Half-black and white symbols represent heterozygotes and wild-type homozygotes, respectively. For a few individuals in the pedigrees, DNA was not available (ND: not determined). PCRs were performed on genomic DNA samples, using 5'- CCTGGCTGTCGTCCATGCTG-3' (SEQ ID NO: 16) and 5'-CTGATCTAGAGCCATGTGCACAACTCT-3' (SEQ ID NO: 17) as forward and reverse primers, respectively. Reaction mixtures consisted in 30 µl of 10 Mm Tris-HCl buffer Ph 8.0, containing 50 Mm KCl, 0.75 Mm $MgCl_2$, 0.2 Mm dCTP, dGTP and dTTP, 0.1 Mm dATP, 0.5 µCi [a-^{32}P]-DATP, 0.01% gelatine, 5% DMSO, 200 ng target DNA, 60 ng of each of the primers and 1.5 U Taq polymerase. PCR conditions were: 93 °C for 2 min 30; 93 °C for 1 min, 60 °C for 1 min, 72 °C for 1 min, 30 cycles; 72 °C for 6 min. After the PCR reaction,

the samples were incubated for 60 min at 37 °C with 10 U *Eco*RI, and 2 µl of the denatured reaction mixture was applied onto a denaturing 5% polyacrylamidc gel containing 35% formamide and 5.6 M urea. Bands were detected by autoradiography. --

Amendments to the Drawings:

The attached 2 sheets of drawings include changes to Figs. 2A and 6B:

Sheet 1, which includes Figs. 2A, replaces the sheet that includes Figs. 2A.

Sheet 2, which includes Figs. 6B, replaces the sheet that includes Figs. 6B.

Attachment: 2 replacement sheet(s); 2 Marked-up sheets showing changes made.